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HASIL PENILAIAN SEJAWAT SEBIDANG ATAU PEER REVIEW  
KARYA ILMIAH : JURNAL ILMIAH**

Judul Karya Ilmiah (Artikel) : Application of Topical Sucralfate and Topical Platelet-Rich Plasma Improves Wound Healing in Diabetic Ulcer Rats Wound Model

Jumlah Penulis : 9 Orang

Status Pengusul : Renni Yuniati,<sup>1</sup> Innelya Innelya,<sup>2</sup> Arti Rachmawati,<sup>2</sup> Harold Jefferson Matthew Charlex,<sup>2</sup> Alfi Rahmatika,<sup>2</sup> Matthew Brian Khrisna,<sup>1</sup> Fârmaditya EP Mundhofir,<sup>3</sup> K Heri Nugroho Hario Seno,<sup>4</sup> Tri Nur Kristina<sup>5</sup>

Identitas Jurnal Ilmiah : a. Nama Jurnal : Journal of Experimental Pharmacology  
b. Nomor ISSN : 1179-1454  
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d. Edisi : Agustus 2021  
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h. Alamat web jurnal : <https://www.dovepress.com/getfile.php?fileID=72655>  
i. Terindeks di : Scopus Q3, SJR 0,56  
j. On line turnitin : <https://doc-pak.undip.ac.id/7655/1/Turnitin-Application-of-Topical-Sucralfate.pdf>

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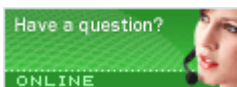
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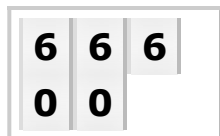
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[Journal of Experimental Pharmacology 2021](#), 13:797-806

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[Journal of Experimental Pharmacology 2021](#), 13:781-795

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Deleeuw V, De Clercq A, De Backer J, Sips P

[Journal of Experimental Pharmacology 2021](#), 13:755-779

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# Experimental Pharmacological Management of Psoriasis

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**Abstract:** Psoriasis is a chronic, relapsing, immune-mediated systemic disease. Its pathogenesis is complex and not fully understood yet. Genetic and epigenetic factors interact with molecular pathways involving TNF- $\alpha$ , IL-23/IL-17 axis, and peculiar cytokines, as IL-36 or phosphodiesterase 4. This review discusses the mechanisms involved in the development of the disease, as well as the therapeutic options proposed following the investigation of the inflammatory psoriatic pathways. We performed a comprehensive search using the words “psoriasis” and the newest molecules currently under investigation and approval. From these data, a new scenario in psoriasis is occurring to personalize the therapies - especially systemic ones and those using small molecules - and avoid topical and injectable drugs. We reported the newest therapeutic opportunities, including the inhibitors of Janus kinase/tyrosine kinase 2, phosphodiesterase-4 and IL-36 receptor. Today, more than 20 molecules are under investigation for the treatment of cutaneous psoriasis. Most of them are constituted by small molecules or biologic therapies. This underlines how psoriasis needs systemic therapies, due to its complex pathogenesis and multisystemic involvement.

**Keywords:** psoriasis, janus kinase inhibitors, tyrosine kinase 2 inhibitors, phosphodiesterase 4 inhibitors, IL-36 receptors inhibitors

## Introduction

Psoriasis is a chronic, relapsing, immune-mediated disease, with a prevalence of 2–3% worldwide. It is characterized by well-circumscribed erythematous plaques, covered by a squamous scale, generally located on the skin of extensor surfaces of the body. 20% of patients with psoriasis may develop a seronegative polyarthropathy - often associated with nail involvement - with a severe quality of life impairment.<sup>1</sup> There are several clinical variants of psoriasis. Besides the psoriasis vulgaris, also called plaque-type psoriasis, we classify guttate, erythrodermic, seborrheic, generalized pustular (GPP) and palm-plantar forms, and, rarely, psoriasis of the mucous membranes.<sup>1</sup> Actually, these clinical variants show different cytokines involved in their pathogenesis and different response to therapies. As psoriasis is a dynamic process, microscopic features vary along the evolution of cutaneous lesions. The earliest lesions could be misinterpreted as they consist in superficial dermis capillary vessels dilatation and mild perivascular lymphocytic inflammatory infiltration.<sup>2</sup> It is not possible to predict the duration of this phase, but it is inevitably followed by the development of the classic psoriatic plaque, which corresponds histologically to confluent parakeratosis with neutrophilic exocytosis in the epidermis. These changes are accompanied by attenuation

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# Pharmacological Modulation of Ion Channels for the Treatment of Cystic Fibrosis

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 Margarida D Amaral<sup>1</sup>  
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\*These authors contributed equally to this work.

**Abstract:** Cystic fibrosis (CF) is a life-shortening monogenic disease caused by mutations in the gene encoding the CF transmembrane conductance regulator (CFTR) protein, an anion channel that transports chloride and bicarbonate across epithelia. Despite clinical progress in delaying disease progression with symptomatic therapies, these individuals still develop various chronic complications in lungs and other organs, which significantly restricts their life expectancy and quality of life. The development of high-throughput assays to screen drug-like compound libraries have enabled the discovery of highly effective CFTR modulator therapies. These novel therapies target the primary defect underlying CF and are now approved for clinical use for individuals with specific CF genotypes. However, the clinically approved modulators only partially reverse CFTR dysfunction and there is still a considerable number of individuals with CF carrying rare CFTR mutations who remain without any effective CFTR modulator therapy. Accordingly, additional efforts have been pursued to identify novel and more potent CFTR modulators that may benefit a larger CF population. The use of *ex vivo* individual-derived specimens has also become a powerful tool to evaluate novel drugs and predict their effectiveness in a personalized medicine approach. In addition to CFTR modulators, pro-drugs aiming at modulating alternative ion channels/transporters are under development to compensate for the lack of CFTR function. These therapies may restore normal mucociliary clearance through a mutation-agnostic approach (ie, independent of CFTR mutation) and include inhibitors of the epithelial sodium channel (ENaC), modulators of the calcium-activated channel transmembrane 16A (TMEM16, or anoctamin 1) or of the solute carrier family 26A member 9 (SLC26A9), and anionophores. The present review focuses on recent progress and challenges for the development of ion channel/transporter-modulating drugs for the treatment of CF.

**Keywords:** anionophores, CFTR modulators, drug development, ENaC, precision medicine, SLC26A9, TMEM16A

## Introduction

Mutations in the gene encoding the cystic fibrosis (CF) transmembrane conductance regulator (CFTR) protein cause CF – one of the most common life-shortening autosomal recessive diseases.<sup>1–3</sup> CFTR is a member of the ATP-binding cassette (ABC) transporter family and functions as a chloride (Cl<sup>-</sup>) and bicarbonate (HCO<sub>3</sub><sup>-</sup>) channel expressed at the apical plasma membrane (PM) of epithelial cells in the airways, intestine, pancreas, sweat glands and other organs.<sup>4,5</sup> This protein is composed of 1480 amino acid residues that are organized into five domains (Figure 1):<sup>6,7</sup> two transmembrane domains (TMD1 and TMD2), two nucleotide binding-domains (NBD1 and NBD2) and an intrinsically disordered

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